AMENDMENTS TO THE SPECIFICATION

Please amend the paragraph beginning on page 1 line 5, as follows:

This application is a continuation of prior application No. 09/232,878, filed January 15, 1999, **now Patent No. 6,245,332**.

Please amend the paragraph beginning on line 3 of page 34, as follows:

Methods are provided to specifically modulate the trafficking of systemic memory T cells, particularly CD4+ T cells, without affecting naive T cells or intestinal memory T cells. It is shown that systemic memory T cells, which are characterized as $CD45Ra^-$, and integrin $\alpha4\beta7^-$, express high levels of CCR4. Ligands of CCR4, such as TARC or MDC, act as an adhesion trigger, wherein upon CCR4 binding, these cells undergo integrin-dependent arrest to the appropriate vascular receptor(s). This arrest acts to localize the cells at the target site. The methods of the invention manipulate this triggering, and CCR4 mediated chemotaxis, to affect the localization of T cells in targeted tissues. ~~**In one embodiment of the invention, the active agent is a CCR4 agonist, that acts to enhance T cell localization.**~~ In an alternative embodiment, the agent is an antagonist that blocks CCR4 biological activity. An advantage of the invention is the selectivity for systemic memory T cells, without affecting native T cells or intestinal memory T cells.

Please amend the paragraph on page 8 line 15, as follows:

Some memory T cells associated with the skin are known to express CLA, and such cells are of particular interest for treatment with the present methods, particularly to modulate the trafficking, or homing of these cells to cutaneous tissues. Conditions of inflammation-associated or allergic reaction patterns of the skin include atopic dermatitis or infantile eczema; contact dermatitis, psoriasis, lichen planus; hypersensitivity or destructive responses to infectious agents, etc. Such diseases benefit from the administration of CCR4 ~~agonists~~ **antagonists**. The treatment decreases the number of systemic memory T cells at the sites of inflammation.